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Review Article

Glaucoma in Penetrating Keratoplasty

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Abstract

Glaucoma following penetrating keratoplasty (PKP) is one of the most common causes for irreversible visual loss [1] and the second leading cause for graft failure after rejection [2]. The management of penetrating keratoplasty and glaucoma remains controversial mainly because of the high risk of graft failure associated with treatment.

Keywords: Penetrating Keratoplasty (PKP); Glaucoma; IOP

Introduction

Penetrating keratoplasty is complicated by a significant incidence of IOP elevation in both the early and late postoperative periods, although reported incidences vary considerably. One study revealed a 31% incidence of early post-operative increase in IOP and a 29% incidence of late (> 3 months) increase [3]. Another large survey had a 9% incidence of immediate postoperative glaucoma and an 18% incidence of chronic post-keratoplasty glaucoma [1]. It was also found that chronic glaucoma was more likely to occur in eyes that had been reported to have early postoperative pressure rise [4].

Associated factors

Factors associated with glaucoma after penetrating keratoplasty in various studies include [1,3-8]:

- Recipient age older than 60 years
- Aphakia
- Pre-existing glaucoma
- Preoperative diagnosis of adherent leukoma
- Bullous keratopathy
- Herpetic keratitis
- Trauma
- Associated vitrectomy
- Anterior segment reconstruction.

The average maximum pressure reported in a study within the first week was 24 mm Hg in phakic eyes, 40 mm Hg in aphakic eyes, and 50 mm Hg in eyes that had combined cataract extraction and keratoplasty [9]. When keratoplasty was combined with cataract extraction, the incidence of glaucoma was higher with intracapsular extraction than with extracapsular surgery [10].

The incidence of post-keratoplasty glaucoma is also increased after repeated penetrating keratoplasty [11].

Pathophysiology

Early postoperative period

In some cases, the postoperative glaucoma after penetrating keratoplasty has similar IOP elevating mechanisms that are associated with other intraocular procedures including uveitis, hemorrhage, pupillary block, and steroid-induced glaucoma [12]. How-

ever, additional mechanisms of early postoperative glaucoma are unique to eyes that have undergone penetrating keratoplasty, especially when aphakia is also present. Two such mechanisms have been postulated.

Collapse of the trabecular meshwork: Collapse of the trabecular meshwork may result from the loss of anterior support due to the incision in the Descemet membrane, which may be compounded in the aphakic eye by a reduction in posterior support from the loss of zonular tension [13,14]. This hypothesis is supported by the observation that full thickness suturing in one study was associated with better aqueous outflow in autopsy eyes and lower early postoperative IOP, compared with conventional suturing [13,14]. Some surgeons, however, report less postoperative pressure rise with use of partial thickness sutures, which they believe prevents angle distortion [15].

Compression of the anterior chamber angle: Compression of the anterior chamber angle may be caused by the conventional techniques of penetrating keratoplasty, causing an early postoperative IOP rise and subsequent chronic glaucoma due to peripheral anterior synechiae [15,16].

Late postoperative period

Gradual flattening of the anterior chamber several months after aphakic keratoplasty has been reported [17]. This appears to be related to presence of an intact anterior vitreous face, and prophylactic vitrectomy has been suggested to avoid this complication. IOP elevation may also occur in association with graft rejection, which may require long-term steroid and antiglaucoma therapy [18]. Pigment dispersion syndrome may also be seen with pseudophakic eyes that have undergone corneal transplantation. In these patients, the syndrome has the unique feature of an inferior linear pigmented endothelial line, which can be confused with graft reaction [19]. Another late-developing glaucoma occurs after keratoplasty for congenitally opaque corneas [20]. It is not associated with peripheral anterior synechiae and the mechanism is unknown. Other forms of late-onset glaucoma may result from peripheral anterior synechiae, the long-term use of steroids, or epithelial ingrowth [12,21].

Diagnosis

Accurate measurement of IOP, assessment of visual fields and neuroretinal structures are often not possible before PK due to the primary corneal disease. This often leads to an inability to diagnose pre-existing glaucomatous optic neuropathy. Following PK, changes in corneal thickness, post-operative astigmatism and refractive changes often preclude reliable post- operative assessment of IOP, disc and visual field.

The diagnosis of post-PK glaucoma is primarily based on IOP measurements in the early post-operative period, and on IOP, optic disc change and progressive visual field changes in the late post-operative period. IOP in the early post- operative period, when the corneal surface is irregular, can be measured with the Mackay-Marg electronic applanation tonometer, the pneumatic applanation tonometer, the tono-pen, or recently the dynamic contour tonometer (DCT), independent of the corneal thickness. If the graft surface is smooth with an intact epithelium and regular mires can be obtained, then Goldmann applanation can be used to measure the IOP.

Management

Preventive measures

Angle compression can be minimized and trabecular support improved by employing following strategies:

- Donor graft that is larger than the recipient trephine
- Looser or shorter suture bites to minimize tissue compression
- Smaller trephine size
- Thinner peripheral host cornea
- Larger host corneal diameter [16,22].

Reports conflict regarding whether an oversized corneal donor graft improves outflow and reduces postkeratoplasty glaucoma. A perfusion study with autopsy eyes revealed no improvement in outflow, and the use of 0.5-mm oversized grafts in a clinical series afforded no protection against postoperative glaucoma [23,24]. However, other clinical studies indicate that the use of oversized grafts is associated with deeper anterior chamber depths, a lower incidence of progressive angle closure, and significantly lower postoperative pressures, compared with use of same-sized grafts [25-28]. Oversized grafts, however, are contraindicated in treating keratoconus because they cause a significant increase in myopia [29]. Another technique to prevent postkeratoplasty angle-closure glaucoma is the placement of sutures near the pupillary portion of a flaccid iris to create a taut iris [30]. In addition, glaucoma after

keratoplasty can be minimized by using meticulous wound closure and extensive postoperative steroids [31] (with caution for steroid responsive patients).

Treatment of glaucoma

Medical therapy

Medical therapy should be tried first, unless a specific, treatable condition, such as pupillary block, is apparent. However, attempts to alter the early postoperative pressure rise are frequently unsuccessful. Carbonic anhydrase inhibitors were not found to be significantly efficacious in this situation [32], although they may be useful in treating the chronic glaucoma. Reported results with timolol have been conflicting [32], although the drug does appear to have some value, especially in controlling chronic glaucoma after keratoplasty [12]. Miotics may occasionally be of value.

Surgical therapy

Surgical therapy is indicated when the optic nerve head or the graft is threatened by a persistently elevated IOP. No glaucoma operation has been found to be entirely suitable for controlling IOP and preserving graft clarity. One investigation found a 30% incidence of graft failure after any intraocular procedure [33]. When penetrating keratoplasty was performed after trabeculectomy in one series, the 5-year probability of successfully maintaining IOP control and a clear graft was only 27%, which increased to 50% in another series of combined trabeculectomy and penetrating keratoplasty [34]. Implantation of a Molteno drainage device achieved IOP control of 21 mm Hg or less with one or more procedures in a series of 17 eyes, although seven had allograft rejections [35]. In another series, involving 26 eyes with glaucoma drainage devices, final IOP was less than 18 mm Hg in 96% of the eyes but graft failure occurred in 42% [36]. Cyclocryotherapy was once the most commonly used surgical procedure for glaucoma after penetrating keratoplasty [37], although the high incidence of serious complications limits its usefulness. Transscleral cyclophotocoagulation has largely replaced cyclocryotherapy as the cyclodestructive procedure of choice. However, in one series of 39 patients, 77% had a final IOP between 7 and 21 mm Hg, but 44% of those with clear grafts before cyclophotocoagulation had graft decompensation [38].

Conclusion

Raised IOP post keratoplasty leads to poor vision and ultimately graft failure. So it is very essential to measure IOP on regular basis. Prompt steps to be taken if found high and should be managed aggressively which can save the graft as well as eye.

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